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Paroxysmal Tachycardia in Newborns and Infants

**A Report of Six Cases Including Intrauterine
Tachycardia, W-P-W Syndrome, and Chronic
Ectopic Atrial Tachycardia**

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Paroxysmal tachycardia (pt) in newborns and infants is not common. In the United States the yearly admission rate has been approximately two per hospital (1); the average physician seeing one case in five years. Paroxysmal tachycardia in newborns and infants, if recognized and treated early, is usually not a fatal illness as cases reported to present indicate.

There are two types of pt: the supraventricular and the ventricular type (1). The supraventricular type shows a perfectly steady heart rate, whereas the ventricular type discloses variations in rate. Supraventricular pt is influenced by reflex vagal stimulation while paroxysmal tachycardia of ventricular origin shows no changes.

This "simple" paroxysmal tachycardia can be successfully treated in almost every instance. However, care should of course be taken to determine whether the tachycardia is of the supraventricular or ventricular type in origin. The initial treatment of supraventricular tachycardia is reflex vagal stimulation. This is especially helpful for paroxysms of short duration. Secondly, drug therapy is considered, with Digitalis being the drug of choice. The treatment of ventricular tachycardia, on the other hand, is entirely

with medication (2). Quinidine and Pronestyl are considered the drugs of choice. In recent years, the use of direct current has been advocated as a successful method of abolishing both ventricular and supraventricular rhythm disorders (3).

With reference to the paroxysms, there are the simple type of tachycardia, as mentioned above, and the repetitive tachycardia (4), a transition between extrasystole and paroxysmal tachycardia. The latter presents repetitive premature beats continuing over months or years, occurring in short bursts or lasting several minutes, but always interspaced by periods of normal sinus rhythm.

A closely similar form of pt has been called chronic ectopic atrial tachycardia (1). The symptoms of this atypical paroxysmal tachycardia showed that; 1) the patient usually has tachycardia lasting for months or years, 2) there are significant differences in frequency while sleeping and awake, without a changing of the EKG, 3) Digitalis frequently produces a-v block without a decrease of the atrial frequency, and 4) the therapy often influences only the a-v block while the pacemaker is not affected at all. Although most cases affect the atrial rhythm, cases of repetitive ventricular pt with ectopic focus have been reported (5). Repetitive and chronic ectopic pt are in some ways similar. However, in chronic ectopic paroxysmal tachycardia, most patients show no normal sinus rhythm intervals as are seen in the repetitive form.

It is the purpose of this report to discuss further cases of pediatric paroxysmal tachycardia in order to possibly obtain a better understanding of this disease.

Case reports

There have been six definite cases of paroxysmal tachycardia at the Universitäts-Kinderklinik in Basel, Switzerland, in the past nine years. The important features of these cases are summarized in the following table. The first two cases are typical cases of pt of the supraventricular type and therefore need no further comment. The remaining four will be more fully discussed as they present some rather unusual and interesting features.

Case No. 3 was a five-month-old white female who, besides having a cold at 4 weeks, was healthy until three days prior to admission. At that time, the patient developed a febrile coryzal infection with accompanying cough.

Age — Sex	Case 1 CZ	Case 2 RL	Case 3 CK	Case 4 RT	Case 5 JG	Case 6 BL
Father	3-week-old white ♂	10-day-old white ♂	5-month-old white ♀	1-day-old white ♂	6-month-old white ♂	1-year-old white ♂
Mother	Healthy	Healthy	Healthy	Healthy	Healthy	Unknown
Pregnancy	Afebrile throat ache 2 weeks prior to term	Without difficulties	Nausea + vomiting during entire pregnancy	Obese, 94 kg	Without difficulties	Without difficulties
Parturition	Hyperemesis	Early, 7 days pre-term, 2800 gm	Late, 10 days post-term, 4050 gm	Frequent emesis + colds induced, uncomplicated, 3,870 gm	Uncomplicated, 3,100 mg	Uncomplicated, 3,140 gm
Infant history	Uncomplicated, 3500 gm	Healthy until 1 day prior to admission. Blue-grey color with dyspnoe. Tachycardia (215/min) for short period	Had cold at 4 weeks. Healthy until 3 days prior to admission, cough, febrile → unconscious for 5 min 2 days prior to admission. Dyspnoe, emesis, unconscious on day of admittance	(Tachycardia detected intra-uterine)	Enteritis at 4 weeks, right inguinal hernia noted at age 6 months	Cough and febrile infection with otitis 11 days prior to admission. Tachy.: 220. Initial therapy: Chloromycin®, treupel®, otrivcin® + leucomycin® nose-drops. Fever reduced but tachycardia persisted 8,800 gm
Admitting Wt.	3600 gm	3100 gm	7,000 gm	3,900 gm	6,900 gm	8,800 gm
General condition	Fair	Good	Poor	Good	Good	Good
Heart	200/min. Auscultated	230/min. Auscultated	No murmurs. Gallop rhythm Present	No murmurs. Gallop rhythm Present	270/min. No murmurs	200/min. Auscultated. No insufficiency
Signs of Insuff. EKG	None	None	Frequency of 330, right axis deviation	None	None	None
X-ray	Frequency of 300, right axis deviation	Frequency of 300, right axis deviation	None taken	Frequency of 140, QRS in middle axis, P increased in lead II	Frequency of 275, QRS in middle axis	Frequency of 220, right axis deviation
Other	Slightly enlarged heart shadow. Density in lower right thorax	Significantly enlarged heart shadow. Slight density in lower right lung field	Respiration increased. Right extremities in extension	Heart shadow negative, thymus hyperplasia, slight shadow in left apical lung	Slight enlargement of right atrium. Thymus hyperplasia. Lungs are negative	Heart shadow negative, lungs negative
	Respiration increased ENT was negative				Skin-circular efflorescence on thorax (rt. anterior)	Oral cavity inflamed
Cause of admission	Collapse and tachy.	Idiopath. Tachy.	Heart insuff. + Tachy.	Tachy. in-utero	Idiopath. tachycardia	Idiopath. tachycardia
Therapy	Prostigmin (0.1 mg)	Cedilanid (4 drops/day)	Reflex vagal stimulation Cedilanid (0.07 mg) Adrenalin (1:1,000)	See discussion	Cedilanid (0.5 cc)	Cedilanid saturation, achromycin, reflex stim.
Response	Pulse decreased to 140/min	Pulse decreased to 180/min	No response		Pulse decreased to 160/min	No response
Progress	Jan. 16: Lung, liver, spleen, and heart are negative. Unexplainable hyperkalaemia treated with glucose IV Feb. 6: No tachy., no murmurs	May 11: No cyanosis, no dyspnoe June 10: Slight tachy. of 160/min present	Aug. 20: Tachypnoic and cyanotic. Digitalization + reflex stimulation without effect. Pronestyl i.v. → bradycardia + asystole. Adrenalin intracardial → heart action → bradycardia. Adrenalin + artif. respirat. without effect → expired	Post-partum showed slight tachycardia Nov. 29: Icterus noted, penicillin + cedilanid were administered Dec. 17: Pulse slow, doing fine	July 25: Reflex vagal stimulation unsuccessful. EKG showed W-P-W signs July 31: Released with digitalization therapy Aug. 26: Readmitted for hernia repair, heart: 160/min Sept. 2: W-P-W noted	See discussion
Final diagnosis	Hyperkalaemia with a (possible resulting) paroxysmal tachycardia	Idiopath. paroxysmal tachycardia	Paroxysmal tachy., heart insufficiency, myocarditis, Coxsackie virus B., atrial septal defect	Intra-uterine tachy., atelectasis	Wolff-Parkinson-White syndrome with resulting paroxysmal tachy. Right inguinal hernia	Chronic ectopic atrial tachycardia

This progressed in two days to dyspnoe, emesis and unconsciousness. A paroxysmal tachycardia was also noted. Reflex vagal stimulation and Digitalization were unsuccessful. 50 mg of Pronestyl was administered i.v. upon which bradycardia and cardiac arrest ensued. Intubation and external cardiac massage were without effect. Upon intracardial injection of 0.5 cc 1 : 1,000 Adrenalin, heart action recommenced but was soon followed again by bradycardia. Intracardial Adrenalin (1.0 cc) and artificial respiration were unsuccessful. The patient expired 3 hours after admission.

Autopsy revealed an atrial septum defect of the foramen secundum type. The heart itself was strongly dilated and showed fatty infiltration of the myocard. There was also fatty infiltration of the liver. The lungs showed atelectasis. Coxsackie Virus B₅ was discovered in the lung and myocardial precipitates.

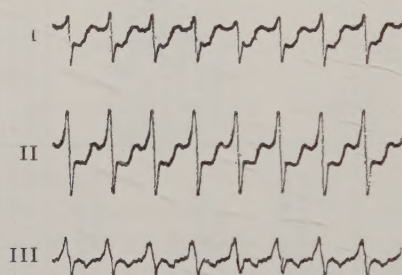


Fig. 1. Case 3. C. K. 5 months old, supraventricular paroxysmal tachycardia. Frequency 316 per min. The P-waves can not be identified but the fact that the QRS-complex is not widened excludes a ventricular origine. There is a ST-T depression due to myocardial anoxia.

Paper speed: 50 mm/sec. 1 mm = 0.1 mV.

Case No. 4 concerns a one-day-old white male. The family history was unremarkable except that the mother was obese (94 kg). The administration of Itinerol-B₆ supp. during the pregnancy was necessary due to frequent emesis.

In June, five months prior to term, and again in the third, second and twice in the last month preterm, the mother was successfully treated for coryzal infections and accompanying sinusitis with Naphthazolin and Hydrocortison nosedrops, and sulfonamides. One month before birth it was first noted that the foetal heart rate was increased to 170 beats per minute. Two weeks prior to term date, the mother again contracted a cold with sinusitis and rhinitis. The foetal heart rate showed no significant change. Eleven days later, the mother was admitted to the hospital with uterine contractions. The foetal heart rate at time of admittance was calculated at 260 beats per minute.

Mild Stroganoff therapy with morphine and chloral to the mother produced partial normalizing of the foetal rate, which, however, increased again to 240 per minute. This increased rate remained until partuition four days later. The

induced birth was uncomplicated. Birthweight was 3,870 g. The birth revealed the umbilical cord to be wound twice around the baby's neck. Oxygen was administered. An EKG showed sinus rhythm, P increased in lead II and a flattened T wave. A chest X-ray revealed a left apical atelectasis with slightly enlarged thymus shadow toward the left. The puls was regular at 140. The blood picture was in the normal range.

Although the heart rate was decreased after birth, Micoren (15 mg), Penicillin ($6 \times 40,000$ U.), and Cedilanid® (0.07 mg) were administered. Three days post-partum, the heart rate was reduced to 128, and all therapy was stopped one week later. There was continued puls quiescence ten days after discontinuing therapy and a repeat X-ray showed no abnormalities. The infant was discharged one month post-partum with no further therapy. The final diagnosis was an intra-uterine tachycardia of unknown etiology along with temporary atelectasis.

Case No. 5 concerns a six-month-old white male who was admitted to the hospital on July 25, 1963. The family history revealed both parents to be in good health with no known illnesses. The mother, a primipara, had an uneventful pregnancy and her delivery was uncomplicated. The baby weighed 3,100 g at birth.

At the age of four weeks, the infant had had an enteritis of short duration. At the age of six months, a right inguinal hernia was first noticed and the patient was admitted for surgical repair. Admitting examination disclosed a generally healthy and well developed child. A tachycardia of about 275 per minute was discovered but no murmurs were noted. A chest X-ray showed a slightly enlarged right atrium. A right inguinal hernia was palpated. Admitting EKG displayed a supraventricular tachycardia.

Bulbus- and carotid sinus reflex stimulation were unsuccessful. At 6 p.m., 0.1 mg Cedilanid was administered i.v. and at 6:30 p.m., sinus rhythm was noted. Cedilanid was thus continued. No vitium, myocarditis, nor other infections were discovered. After six days, a control EKG disclosed a strong possibility of a Wolff-Parkinson-White (W-P-W) Syndrome (6). The infant was discharged from the hospital under digitalization for control of the pt until and including the operation of the hernia.

One month after initial admittance, the patient re-entered the hospital. The heart frequency was 160 per minute and no murmurs were auscultated. The following day, an uncomplicated herniotomy after Girard was performed. One week post op., a repeat EKG showed definite signs of W-P-W Syndrome. A repeat X-ray, except for an enlarged thymus shadow, was negative. The patient was discharged with a limited supply of Cedilanid (0.1 mg per day). Final Diagnosis was W-P-W Syndrome and paroxysmal tachycardia along with a right inguinal hernia.

Case No. 6, a one-year-old white male, was admitted to the Children's Hospital on December 9, 1963. The family history revealed that the health of the mother had always been good. The father was unknown. The pregnancy was without maternal illness and the mother, a primipara, had an uncomplicated delivery. The baby weighed 3,140 g at birth.

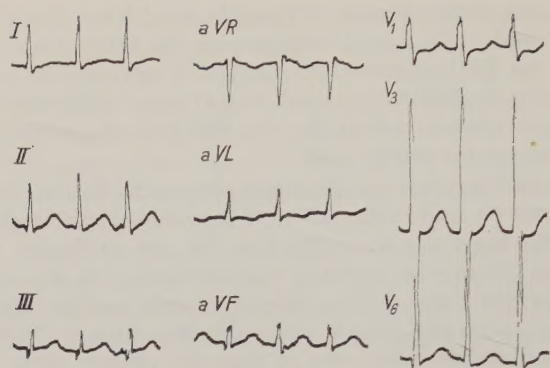


Fig. 2a

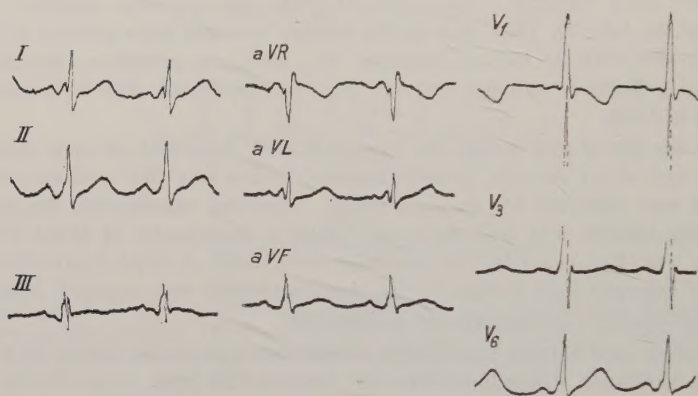


Fig. 2b

Fig. 2. a) Case 5. J. G. 6 months old. Supraventricular paroxysmal tachycardia. Frequency 275 per min. P-waves not visible. No ST and T wave changes. b) After normalization of the heart rate (frequency 136) the typical pattern of the Wolff-Parkinson-White syndrome appears: shortening of P-Q, wide QRS with a Δ -wave in standard lead I, in aVL and V6.

Paper speed: 50 mm/sec. 1 mm = 0.1 mV.

Eleven days prior to admission, the child was noticed to be very restless with a febrile infection, cough, and accompanying otitis. A tachycardia was discovered (220/Min.) along with gallop rhythm. Initial therapy administered by the family physician was Achromycin®, Treupel® supp., Otrivin® along with Leukomycin® eardrops. The baby's fever subsided but the heart rate continued around 180 beats per minute.

On admission, the child appeared slightly dystrophic with no noticeable cyanosis or edema. The oral cavity was inflamed, and the respiratory frequency was increased. An EKG showed sinus rhythm with a rate of 200 per

minute and a negative P wave in D₂ and normal QRS complex. No heart insufficiency was noted. A chest X-ray revealed the heart shadow to be within the normal limits. The patient was given a saturation dose of Cedilanid®, 0.24 mg/24 hrs., plus Achromycin®. The following day it was noted that the puls remained around 200 per minute whenever the baby was crying, and decreased to around 150 when he was quiet.

On the second hospital day, 3×0.05 mg Reserpin® was administered for sedative effect. Vagus stimulation through reflex bulb pressure was unsuccessful. Due to the unsatisfactory response to Cedilanid®, it was believed that a myocarditis was possibly present along with the tachycardia. Two days later the heart frequency was still unchanged. Upon administration of 175 mg Chinidin, a partial a-v block with a noticeable decrease of frequency occurred, but unfortunately with no satisfactory result. Therefore, first Chinidin was stopped and then Cedilanid® was reduced. One month after admittance, the heart frequency was 150-160 per minute and Cedilanid® therapy was stopped as it appeared to have little effect.

On February 15, nine weeks after admittance, it was decided to administer a combination therapy consisting of digitalization, sedation, and sympathicolysis. When 1.0 mg Dihydroergotamin (DHE) 0.1 ml Cedilanid® and 45 mg Luminal were administered, a regular puls of 100 per minute with a normal EKG picture was realized. This quiescence was still noted on February 27, and Cedilanid® was stopped 12 days later. No change was seen in the frequency. Finally, all therapy was discontinued and the patient received only vitamins. The heart frequency remained at 100 per minute and the infant was released from the hospital fourteen weeks from the date of admittance. Final Diagnosis was a chronic ectopic atrial tachycardia.

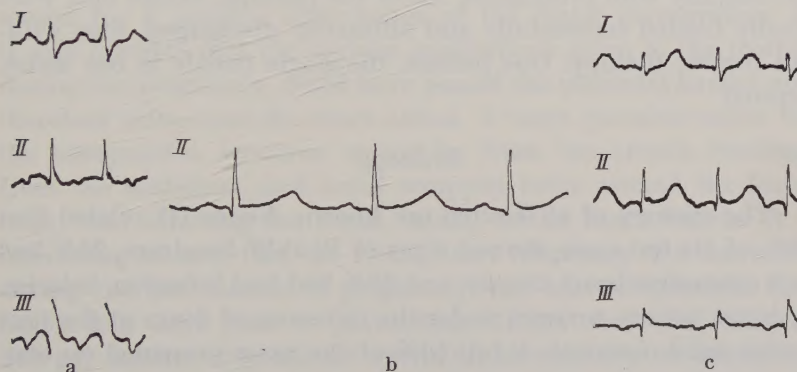


Fig. 3. a) Case 6. B. L. 12 months old. Supraventricular paroxysmal tachycardia. Frequency 260 per min. b) Normal ECG during sleep with a frequency of 120 per min. c) ECG 4 months after discharge from hospital. Frequency 187. Negative P-wave in standard lead II suggesting an ectopical pacemaker.

Clinically the infant is without symptoms.

Paper speed: 50 mm/sec. 1 mm = 0.1 mV.

Comment

There have been six cases of pt in the past nine years at the Children's Hospital in Basel, Switzerland. It is interesting to note that *all occurred in infants less than one year of age*. This agrees with the literature (1) in that the disease usually begins within the first four months of life. With infants (until the fourth month) generally no etiological factors are found, whereas older infants and children frequently show reasons which might explain the occurrence of the tachycardia, e.g. W-P-W, congenital heart disease, infections, etc. (7).

The presented cases concerned five males and one female. The families of these children were generally healthy. The mother's pregnancies in cases 1, 3, and 4 were complicated with hyperemesis. The mother of case 4 also had had frequent colds during her pregnancy. The deliveries in all cases, however, were uncomplicated. The infants in cases 2 and 6 had had coryzal infections, and case 5 had had enteritis prior to the tachycardia.

Except for the tachycardia in utero, all cases showed an increased frequency of at least 200 per minute or more at the time of admission. In five of the six cases, no signs of cardiac insufficiency were detected. Cedilanid® was administered as initial therapy to five of the six cases, but a beneficial response was noted in only three of those treated. The remaining case, No. 1, was treated successfully with prostigmin. All of the patients, except one, were finally treated successfully and ultimately discharged, four without further therapy. One patient, the single female in our series, expired.

Etiology

The etiology of pt is often not known. Nadas (1) related that 10% of his test cases showed signs of W-P-W Syndrom, 20% had had congestive heart disease, and 20% had had infection, injuries, or heart tumors, or were under the influence of drugs at the time tachycardia occurred. A full 50% of the cases presented no etiology whatsoever.

The possible etiologies in our six cases are as follows: The mother in Case No. 1 was noted to have had an upper respiratory infection two weeks prior to term. An unexplainable increase in serum potassium (25 mg%) was also noted in the infant post par-

tum. Hyperkaliemia may cause arrhythmia and a widening of the QRS complex. It is dubious, however, that this slight elevation of potassium could have been the cause of the tachycardia, especially when we consider that the potassium value remained increased even after the tachycardia subsided.

Case No. 2 appears as a true idiopathic tachycardia. The infant was well until he suddenly became dyspnoic and cyanotic on the tenth day post-partum. These symptoms lasted for only a few minutes but a tachycardia of 230 per minute remained. This was ultimately controlled with Cedilanid® and the patient was discharged after one month hospital observation, with no further therapy.

In Case No. 3, the tachycardia was believed to be due to an infection of the myocard itself. The quickness of death was believed to be the result of a too quick administration of a therapeutic dose of Pronestyl. This no doubt acted on the myocardium which was itself damaged by either anoxia or by inflammation. It is interesting that Coxsackie Virus B₅ was isolated from the myocardial precipitate. However, histological examination revealed very little inflammation of the heart muscle. Autopsy also showed an atrial septum defect which doubtlessly had an affect on the outcome.

The etiology of Case No. 4 is not clear. Certainly the febrile infections which arose during pregnancy could be considered as a reason for a possible disturbance of the foetal myocardium. It is also possible that the various medications given to the mother during her pregnancy, could have passed the placental barrier and therefore influenced the heart action. A more plausible cause for the tachycardia, however, would be from the anoxia resulting from the umbilical cord being wrapped twice around the foetal neck, thus affecting the blood supply to the heart muscle. It is interesting to note that the foetal heart frequency had increased during the last month of pregnancy, but true tachycardia first began four days prior to parturition. At that time the rate was 240 per minute. The almost spontaneous recovery of the infant post-partum supports anoxia as the cause of the tachycardia in utero.

Case No. 5, a six-month-old white male with no outward symptoms of pt, was admitted to the hospital for hernial repair. The supraventricular tachycardia was discovered during the ad-

mitting examination and therapy was then begun. There was no known etiology; no vitium, no myocarditis, no infections. Only after the tachycardia had been "quieted down" with Cedilanid®, was the W-P-W Syndrome first detected. It appears that during the tachycardia of 275 per minute, the underlying signs of the syndrom were unrecognizable. It is clearly possible that the infant could have had the W-P-W syndrome since birth as a possible congenital manifestation (8) which remained undetected until the pt was discovered upon hospital admittance. It may be of value therefore, to review the EKGs in all pt cases after a successful slowing of the heart rate has been achieved. In this way it may be possible to recognize whether W-P-W Syndrome is the cause of a tachycardia.

In Case No. 6, the paroxysmal tachycardia had no known etiology. The one-year-old white male was perfectly healthy until he contracted a febril coryza along with otitis. A tachycardia of 220 per minute was also found. Whether the infection and otitis played a part in the arising of the tachycardia is open to discussion. Certainly no other causes were noted. This case closely follows the description given of chronic ectopic atrial tachycardia (1). Although the etiology is not known, *Nadas* (1) reports that from the cardiac side, progress throughout the juvenile years is good. It may be worthwhile to treat these patients even though control of the ectopic focus may not be fully realized. Slowing the tachycardia and increasing the a-v block may lead to a decrease of ventricular rate which may in turn prevent the development of congestive heart failure. This was the procedure adopted in our particular case. A combination therapy of sedation, sympathicolysis, and digitalization did indeed finally decrease the tachycardia.

Summary

Six cases of paroxysmal tachycardia in newborns and infants are presented. All six patients had different histories, the only possible similarity in their etiologies being some type of infection in the infant or pregnant mother. Four among these six cases had uncommon and interesting features and are therefore discussed in some detail. Of particular interest are the cases of W-P-W Syndrome, Intrauterine tachycardia, and Chronic Ectopic Atrial Tachycardia.

Zusammenfassung

Es wird über 6 Fälle von paroxysmaler Tachycardie bei Neugeborenen und Säuglingen berichtet. Jeder der 6 Patienten hatte eine andere Krankengeschichte. Lediglich die Tatsache, daß bei den Kindern oder der graviden Mutter irgendeine Infektion bestand, läßt eine gewisse Ähnlichkeit in ätiologischer Hinsicht erkennen. Vier der sechs Fälle wiesen ungewöhnliche und interessante Befunde auf, die eingehender besprochen werden. Von besonderem Interesse sind die Fälle mit WPW-Syndrom, von intrauteriner Tachycardie und von chronischer ektopischer Vorhof-Tachycardie.

Résumé

L'auteur rapporte les observations de 6 cas de tachycardie paroxystique rencontrée chez des nouveau-nés et des nourrissons. A part une parenté étiologique sous forme d'une infection survenue soit pendant la grossesse soit après la naissance, l'histoire de la maladie chez les sujets atteints était différente d'un cas à l'autre. Parmi les six cas, quatre observations inhabituelles et intéressantes ont été exposées d'une façon approfondie, dont le syndrome de W. P. W., la tachycardie intrautérine et la tachycardie auriculaire chronique ectopique.

Resumen

Se presentan seis casos de taquicardia paroxística del recién nacido y lactantes. Los seis pacientes tenían una historia clínica distinta, siendo la única semejanza posible en su etiología algún tipo de infección en el niño o en la madre embarazada. Cuatro de estos seis casos ofrecían particularidades interesantes y no habituales por lo que son descritas con algún detalle. De especial interés son los casos del síndrome de W. P. W., taquicardia intrauterina y taquicardia auricular ectópica crónica.

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